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POTASSIUM AND SODIUM IN PEDIATRIC NUTRITION

By EDWARD L. PRATT, M.D.*

Minerals, although not energy producing, are indispensible to life and must be provided from our food each day. Throughout ancient fable and folklore the "salt of the earth" has been second only to the "water of life." From the point of view of water and electrolyte metabolism, life was at its simplest when a conglomeration of chemicals within a cell wall first maintained continuous metabolism while freely floating in the Pre-Cambrian ocean. In order for our remote ancestors to leave the sea for the land, increasingly complex machinery had to be devised. More and more cells had to accept specialized functions. Vast portals of entry — the gastrointestinal tract and the lungs — had to be established to bring in essential metabolites and oxygen. A transportation system had to be devised, and a competent disposal system evolved. To put the "fine adjustment" on these complex interrelationships of intake, outgo, and internal distribution and exchange, a system of diffusible regulators had to be added — the endocrine glands.

Pediatricians have always played an important role in the development of this phase of medicine because of the great frequency with which infants and children suffer from abnormally large losses or from reduced intake of water and electrolytes. In one of the earliest pediatric texts, in 1727, Mauriceau wrote: "As soon as little infants are in the least indisposed, they very ordinarily get a looseness to which their natural moisture very much contributes." Also the serious disadvantage of small size with relatively large surface area and higher metabolic rate compels a high turnover rate for water and electrolytes in infants and small children. So handicapped, they can survive only briefly experiences of deprivation or increased losses which would scarcely upset an adult.

Practically every physician must have considerable knowledge of electrolyte-physiology because important derangements of it are frequent. Many of the "advances" in present day medicine depend for their maximum effectiveness on the control of the electrolyte abnormalities.

The minerals potassium and sodium are present in ample amounts in practically all foods. Absorption from the gastrointestinal tract for these substances is almost complete, and amounts present in the body in excess of

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need are promptly excreted in the urine. Thus, in health, the principal factors regulating the amounts of sodium and potassium in the body are intake and renal output of these ions. However, the distribution of these ions within the body and their derangements by disease are more complex and will be considered separately, after considering the composition of the body.

Composition of the Body

The composition of the body has been of interest from very early times. The range within which the "true" values for the various body constituents may lie is a pretty broad one due to the inherent variation in all biological material, the observational variation in the methods which must be used, and, particularly, the unobliging features of the body, such as its lack of homogeneity and the varying rates of turnover of various substances. Nevertheless, many of the approximations now available are sufficiently exact to satisfy practical requirements. At present the best compromise is to consider the fluids and electrolytes in relation to the whole body which is made up of fat, skeletal solids, transcellular water and two phases: an intracellular phase consisting of cell solids and cell water, and an extracellular phase composed of connective tissue solids, connective tissue water and the remaining extracellular water (1). An approximation of the body content of water, sodium, potassium and chloride for infants and children may be derived from the equations of Forbes (2). Table I depicts current estimates for the fluid and electrolyte values at birth, one year of age and in adults.

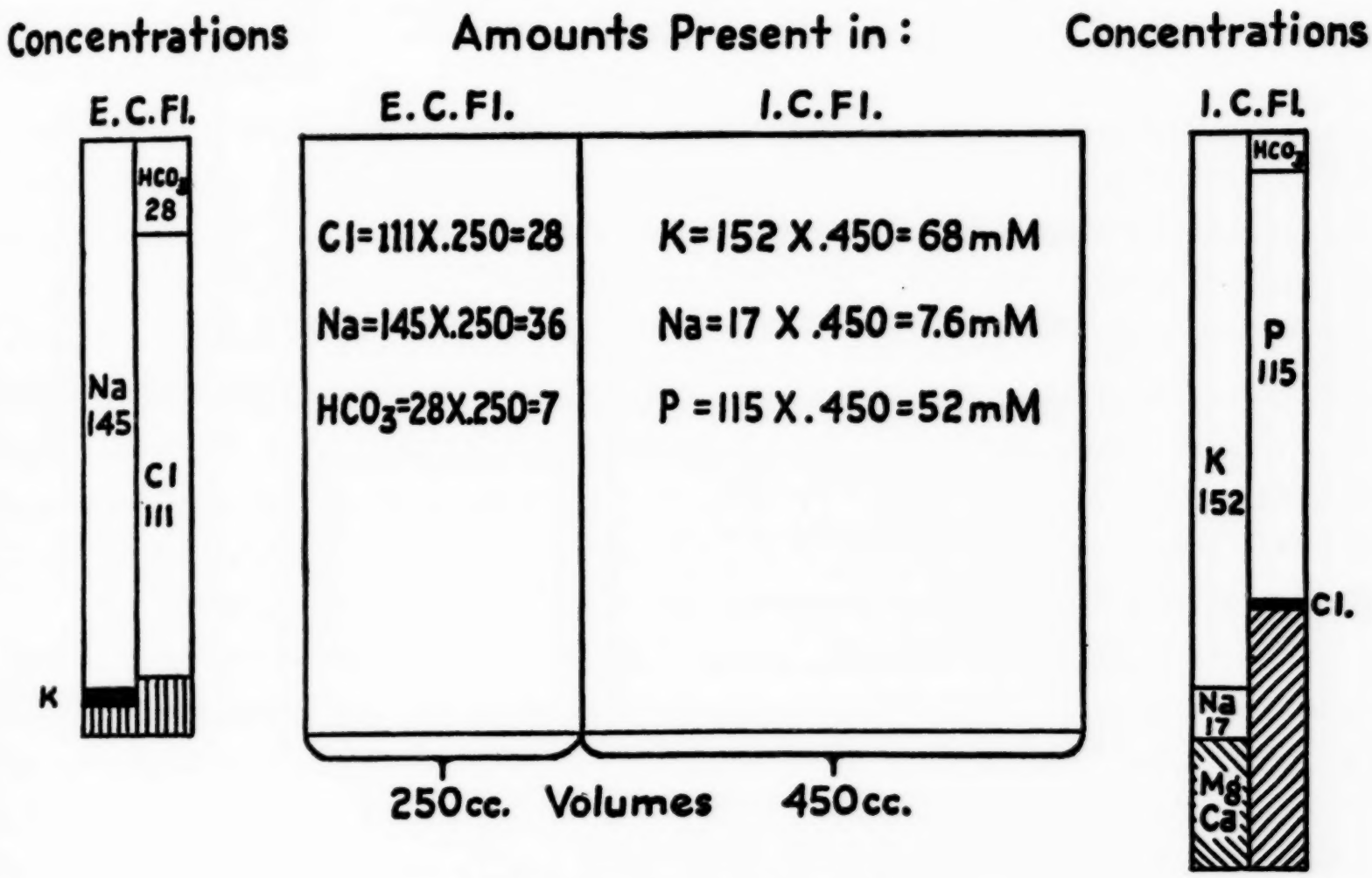
TABLE I. (2)

Total Body Water, Sodium, Chloride and Potassium

			Adult	
	Birth	*One Year*	*Male*	*Female*
Water ml/kg.	726	659	620	515
Sodium meq/kg.	75	62	42	40
Chloride meq/kg.	51	41	32	29
Potassium meq/kg.	42	43	47	41

A gross, but useful, simplification of the anatomy of the water and electrolyte structure of the body in an infant is depicted in Figure I. It is apparent that the principal extracellular cation is sodium, whereas potassium is the major cellular cation. A significant amount of sodium normally resides within the cells and is capable of being reduced to nearly zero or of greatly increasing in value under certain circumstances. Bone contains appreciable quantities of sodium and potassium.

FIGURE I.



Physiological Role of Potassium

It is essential that the cells contain their usual amount of potassium in order to function properly, particularly those cells of the cardiac, smooth or skeletal muscles and those in the central nervous system (3). Abnormal losses of potassium, which may lead to symptoms or death, usually result from one of three mechanisms:

I. Whenever the potassium balance becomes negative due to decreased dietary intake or as the result of abnormal losses from the gastrointestinal tract or from renal dysfunction. Low or zero intake may be produced by anorexia or nausea from illness and is present in those patients maintained on parenteral fluids which contain no potassium. All gastrointestinal fluids contain potassium, and therefore loss of these fluids by vomiting, from diarrhea, or by aspiration of the stomach or upper intestinal tract may produce potassium depletion. In many cases the urinary loss of potassium is a major factor, but, in most instances, such excretion is secondary to other changes. Primary renal disease producing loss of potassium is not frequent.

II. The second mechanism leading to potassium depletion is an inability

of the cells to maintain their high intracellular concentration of potassium due to:

(1) depressed metabolism
(2) hormonal effects
(3) excessive sodium intake

Apparently the high cellular concentration of potassium is dependent upon the energy derived from aerobic oxidation or anaerobic glycolysis (4). Depression of metabolic activity produces a loss of potassium and an increase of sodium within the cells. Adrenocortical hormones, especially aldosterone (5), have a similar influence on the cellular potassium and sodium content. Dehydration, in the presence of appreciable renal function, will lead to a loss of cellular potassium. Finally, the reciprocal relationship between high sodium intake and increased potassium excretion, first noted by Bunge and recently reinvestigated by Gamble (6), constitutes an additional example of the inability of the cells to maintain the usual amounts of potassium.

Thus any situation wherein "shock," dehydration, circulatory insufficiency, or anoxia are present may lead to potassium depletion. Adrenocortical tumors or the administration of ACTH, cortisone or related steroids and the naturally occurring "stress" accompanying illness will tend to decrease cellular potassium. Injudicious use of sodium salts orally or parenterally may also result in the loss of cellular potassium.

III. The third mechanism whereby the amounts of cellular potassium may be altered are by changes in the hydrogen ion concentration of the extracellular fluid. When serum pH is increased (alkalosis) the cells tend to lose potassium and gain sodium. If the serum pH is lowered (acidosis) the cells tend to have an increased amount of potassium and a decreased amount of sodium. Therefore, the intracellular sodium varies directly with the pH of the serum, and the cellular content of potassium varies inversely with the pH of the serum. Conversely, if the primary alteration is within the cells so that there is a lowered cellular potassium, the eventual equilibrium reached is one of elevated serum pH, (metabolic alkalosis).

Table II lists the symptoms and signs most frequently observed in uncomplicated states of potassium deficiency (7).

Important elevations of extracellular potassium, which may lead to death as a result of heart block, occur only from serious impairment of renal function (8) or anuria (9), unless inappropriately large amounts of potassium are given at too rapid a rate parenterally.

Physiological Role of Sodium

Sodium is the major cation of the extracellular fluid and also is the major

TABLE II.

Symptoms and Signs of Potassium Deficiency

Nervous System	
Mental State	Weakness, lethargy Apprenhension Depression, confusion
Reflexes	Depressed
Cardio-Vascular System	
Heart	Tachycardia Sounds of poor quality Enlargement Appearance of murmurs Appearance of congestive failure
Blood Pressure	Widening of pulse pressure Hypotension
Pulse	"Corrigan" pulse Irregular Rhythm
Veins	Elevated venous pressure
Respiration	Shallow, gasping respirations Dyspnea, cyanosis
Gastro-Intestinal Tract	Nausea, vomiting Abdominal distention Paralytic ilens
Muscles	Muscular weakness Soft, flabby, hypotonic muscles Flaccid paralysis
Laboratory Abnormalities	
Electrocardiogram	Lowered, flattened, or inverted T waves U waves Depressed ST segments Extrasystoles, AV block
Serum Concentrations	
pH	elevated
HCO_3	elevated
Cl	decreased
K	lowered in presence of good renal function May be normal or high
Serum Proteins	May be lowered
Glucose	Hyperglycemia or decreased glucose tolerance may be present

factor maintaining the tonicity of the extracellular fluid. Conservation of sodium, when intake is low or if losses from the gastrointestinal tract or skin have occurred, is accomplished by a marked reduction in urinary sodium excretion. Overabundant intake of sodium in healthy persons is adjusted to by increasing urinary excretion of sodium, albeit at a leisurely rate (6). Under ordinary circumstances, the tonicity of the extracellular fluid seems to be controlled by the osmo-receptor system whereby the output of posterior pituitary antidiuretic hormone is altered so as to regulate the renal excretion of water (10). The volume, as well as the tonicity, of the extracellular fluid is highly important to the integrity of the circulation. Therefore, there must be some mechanism sensitive to changes in the volume of the extracellular fluid, and this mechanism may be activated to promote salt and water retention whenever the "functionally effective" circulation is inadequate.

The kidney plays a major role in maintaining the composition and the reaction of the body fluids. The usual diet, the metabolic end products and the intestinal excretions, all require the kidney to excrete more chloride, phosphate, sulfate and organic acids than sodium, potassium, calcium and magnesium. This is accomplished by two ion exchange mechanisms and an interrelated mechanism for ammonia production (11, 12). As a result of these mechanisms, fixed cations are saved, and a urine is excreted that is acid in reaction, contains little bicarbonate, has chiefly monobasic phosphate and has an increased ratio of free organic acids to salts of organic acids. In addition some of the chloride and sulphate ions are covered by ammonium ions. The renal defense against alkalosis is one in which sodium is excreted with bicarbonate and, possibly, also with organic anions, particularly citrate, which may exchange from the cells for chloride from the urine.

In addition to the above renal factors, the urinary excretion of sodium is profoundly altered by the action of certain adrenal hormones, particularly aldosterone (5). These "salt-retaining" compounds from the adrenal cortex lead to a decreased excretion of sodium in the urine and an increased amount of urinary potassium.

Therefore, we see that the maintenance of the proper amounts of sodium and water and the regulation of the concentration of sodium in the extracellular compartment requires a complex integration of the cardiovascular system, the kidneys, the neurohypothysis, the factors controlling blood pressure, and the adrenal and other endocrine glands (3).

In addition, sodium belongs within the cells and can shift from the extracellular fluid into or out of the cells. This shift of sodium from the extracellular to the cellular compartment and vice versa is an important

mechanism for diminishing the variation in the concentration of bicarbonate in the extracellular fluid, and may lead to significant changes in extracellular sodium without any change in the total body sodium.

Clinically the disturbances of sodium of major importance are:

1. Those associated with changes in the *amount* of extracellular sodium.
2. Those with increased or decreased *concentration* of sodium in the extracellular fluid.
3. Those involving *shifts* of sodium into or out of the cells.

Deficits of sodium usually arise, not from curtailed intake, but from abnormal losses. Diarrheal disease and other abnormal losses of gastrointestinal fluids are the most frequent cause of sodium loss in pediatric patients. Sweating may be of considerable magnitude in children and salt losses may be unusually large if the concentrations of sodium and chloride in the sweat are abnormally high as in cystic fibrosis of the pancreas (13). Adrenal insufficiency and diabetes mellitus are examples of endocrine abnormalities leading to loss of sodium. "Salt-losing" types of chronic nephritis may occur in infants and children. Decreases in the total amount of sodium in the extracellular fluid lead to a deterioration of circulatory efficiency which produces a decrease in cerebral and renal functions.

The physiological derangements associated with decreased concentrations of serum sodium, hyponatremia, have received much attention lately. The origins of "hyponatremic syndromes" are complex (14). One type of this syndrome arises in dehydration when salt loss has been proportionately greater than that of water, i.e. "hypotonic dehydration." Abnormal losses of sodium, failure of the elaboration of adrenal "salt-retaining" hormones, excessive use of diuretics, or renal unresponsiveness may be the primary event.

Excessive accumulation of water with little change in total body sodium produces another type of hyponatremia, "sodium dilution." This type occurs when water is given in excess of the patient's ability to excrete it, or when antidiuretic hormone production continues due to pain, after surgical procedures, or because of circulatory insufficiency. Both of these depletion and dilution hyponatremic syndromes are symptomatic and both respond dramatically to the use of hypertonic saline or water deprivation, respectively.

Hyponatremia unassociated with any symptoms has been observed in chronically ill patients, particularly those with tuberculosis, prolonged cardiac failure, liver disease and nephrosis. It is felt that this "symptomless hyponatremia" may be related to decreased intracellular osmolarity (15).

Hypernatremic syndromes have long been recognized. In this disturbance the circulation is relatively well maintained and cerebral cortical functions deteriorate and death results from respiratory failure (16). Thirsting is the

prototype for this disturbance. High salt intakes with insufficient water intake or lossses of hypotonic fluids will lead to hypernatremia.

Associated with increased total body sodium is edema in the absence of grossly overabundant fluid intake or seriously impaired water excretion. In the usual types of edema there is an increase in the extracellular fluid volume so that sodium retention must have been present during its accumulation. Increased capillary hydrostatic pressure, a decrease in the concentration of serum albumin, increased interstitial protein content, or lowered interstitial tissue pressure will upset the hydrostatic-osmotic equilibrium towards increased interstitial fluid volume (17). Decreased absorption and return of interstitial fluid because of impaired lymphatic function is another mechanism. Excessive renal conservation of sodium undoubtedly plays a major role in most instances of edema and ascites. Excessive aldosterone secretion has also been observed in nephrosis, congestive failure, cirrhosis, eclampsia and in postoperative states (5). Therefore, the management of edematous states is first directed at the underlying primary cause and secondly at the metabolism of sodium. Restriction of sodium intake and abstraction of sodium from the body by means of diuretics are frequently employed. Clearly the dangers of sodium depletion must be kept in mind.

Dietary Considerations

There are no stores of dispensable sodium or potassium in the body. Therefore, except in the situation of starvation where the breakdown of body tissue releases these substances, intake must equal outgo each day in the adult. In children, intake must exceed outgo by the small amounts of sodium and potassium required for growth. The allowances for intakes of sodium and potassium, like most food substances, are best viewed from the point of view of minimal requirements and the maximum amounts which can be physiologically tolerated. In health and in the absence of sweating, the minimal requirements for sodium and potassium are largely dependent upon the conservation of these ions by the kidney and the needs for growth. Beyond early infancy the urine can be rendered practically free of sodium and chloride and may contain potassium at no greater concentration than in the blood serum. Thus the minimal urinary losses of these electrolytes are approximately 0.2 milliequivalents of sodium and chloride and 0.4 milliequivalents of potassium per 100 Calories metabolized (3).

The needs for growth are difficult to estimate. From the quite constant composition of the body, except for fat content, as determined by whole body analyses (18) and isotope dilution studies (2) it is evident that the large growth requirements of sodium and potassium calculated by extrapolation

from the positive balances in balance studies must be too large (19). Also the widely different retentions "for growth" for infants fed cow's milk as compared with human milk may well be fallacious (20). The best estimates available for growth in young infants seem to be of the order of: 76 milliequivalents of sodium, 58 milliequivalents of chloride and 46 milliequivalents of potassium per kilogram of body weight gained (19).

Metabolic studies indicate that a moderate amount of sweat is formed under usual environmental circumstances producing an additional loss of about 0.5 milliequivalents of sodium and chloride and about 0.2 milliequivalents of potassium per 100 Calories metabolized.

Fortunately, in planning diets for healthy infants and children these considerations of minimal requirements have little practical significance. The cardinal principle of pediatric nutrition education is to so manage the psychologic aspects of feeding that the eating of a wide variety of natural foodstuffs is a pleasurable experience for the child and his mother. Therefore, all such diets, adequate in other respects, will automatically contain ample quantities of sodium and potassium. Although the sodium, potassium, and chloride figures for human and cows milk cited here (21) are not in complete agreement with the mean values reported in the National Research Council's bulletin No. 119 on the composition of milk, they are certainly representative, and fall within the range of normal values.

Type of Milk	*Potassium*		*Sodium*		*Chloride*	
	meq/L	mgm%	meq/L	mgm%	meq/L	mgm%
Human	10.5	41	4.8	11	9.0	32
Cow's	40.0	155	22.0	50	30.0	105

Thus the young breast fed infant will have an average intake of about: 1.6 milliequivalents (60 mgm) of potassium; 0.8 milliequivalents (16 mgm) of sodium; and 1.3 milliequivalents (48 mgm) of chloride per kilogram of body weight per day. For practical purposes these amounts may be considered as the lower usual allowances for good nutrition in well infants. In teen age children 3 Grams of sodium chloride and 2 Grams of potassium may be considered as the lower usual allowances.

The maximum tolerated amounts of sodium and potassium have not been defined. High intakes of minerals command a large supply of water for urine formation and there is an antagonism between high intakes of sodium and potassium balance, or vice versa, as previously explained. Infants ingesting large amounts (200 ml. per kilogram of body weight) of undiluted cow's milk will be receiving approximately: 8 milliequivalents (310 mgm)

of potassium; 4.4 milliequivalents (100 mgm) of sodium; and 6 milliequivalents (210 mgm) of chloride per kilogram of body weight each day. From a practical point of view abnormally large amounts of sodium and potassium will not be taken in diets ordinarily prescribed for children, except for a few instances where excessive amounts of sodium chloride are added to foods during preparation or at the table.

Special Diets

Special diets with respect to sodium and potassium play a limited role in pediatrics due to the relative infrequency of the diseases for which such special diets are indicated, and also because of the peevish appetites of sick children and the frequency with which children will refuse diets in which the amounts of sodium and potassium have been altered. Most body fluid disturbances in childhood are the result of acute illnesses and demand initial restorative measures by means of parenteral fluid therapy, the intelligent application of which requires a more comprehensive knowledge of water and electrolyte physiology and pathology than it seems desirable to review here. As soon as these patients are able to take their usual caloric intake, a normal diet for age will supply all the needed electrolytes, in most instances.

Low sodium diets, constructed around low sodium milk and low salt foods, may have as little as 2.5 milliequivalents of sodium (about 58 mgm. of sodium or approximately 150 mgm. of NaCl) (22). Low sodium diets are useful in patients with cardiac failure, edema from nephrosis or other causes, and when adrenal steroid therapy is employed.

Increased sodium intake may be prescribed in unusually hot weather, especially for those patients with pancreatic fibrosis or adrenal insufficiency. In the latter instance one to ten grams of added sodium chloride is frequently employed irrespective of the weather. In special types of nephritis, those with limitation of ability to conserve fixed base or sodium, may require a special diet with added amounts of sodium.

A potassium-free diet is of great importance in patients with anuria (9) or severe oliguria, such as in acute glomerulonephritis. Since practically all foods contain potassium, the important feature of the diet prescription for these cases is that no foods except those known by analysis to be free of potassium should be employed during the brief period of renal failure.

The restoration of cellular constituents after deficits have occurred is generally a gradual process. Therefore, potassium is frequently added to diets of infants and children during convalescence from illnesses in which potassium deficiency was present — diarrheal disease, prolonged vomiting, diabetes mellitus, and some postoperative states. Usually potassium, as potas-

sium chloride, is added to the total day's intake of oral fluids or milk in infancy, and as divided doses throughout the 24 hour period in older children. Added potassium is employed until a full caloric intake of the usual foods is being taken.

Finally the combination of a low sodium diet and additional potassium (sodium of from 2.5 to 10 milliequivalents per day and potassium at the level of 150 milliequivalents per day) is often employed in patients being treated with adrenocortical hormones, thereby controlling the tendency towards salt retention, hypertension and potassium deficiency which may complicate therapy with these substances (22).

Summary

The electrolytes sodium and potassium are indispensible items as vital as any element in the diet.

Fortunately simple milk diets in early infancy and all well balanced diets for older infants and children provide suitable amounts of sodium and potassium.

Special diets, with altered amounts of sodium or potassium, play an infrequent, but none the less important role in the management of several serious pediatric illnesses. As our understanding of the physiology and pathology of these ions progresses the field of usefulness of such special diets should be enlarged.

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